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PB-253 677

# Criteria Document for Toxaphene

Environmental Protection Agency

June 1, 1976

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EPA-440/9-76-014

PB 253 677

# CRITERIA DOCUMENT

# TOXAPHENE

REPRODUCED BY
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INFORMATION SERVICE
U.S. DEPARTMENT OF COMMERCE
SPRINGFIELD, VA. 22161

| BIBLIOGRAPHIC DATA SHEET | 1. Report No. EPA-440/9-76-014 | 2. | 3. Recipient's Accession No. |
|---|---|---|---|
| 4. Title and Subtitle<br>Criteria Document for Toxaphene | | | 5. Report Date<br>June 1, 1976<br>6. |
| 7. Author(s)<br>Anonymous | | | 8. Performing Organization Rept. No. |
| 9. Performing Organization Name and Address<br>U. S. Environmental Protection Agency<br>Office of Water Planning and Standards<br>401 M Street, S.W.<br>Washington, D.C. 20460 | | | 10. Project/Task/Work Unit No.<br>11. Contract/Grant No. |
| 12. Sponsoring Organization Name and Address<br>Office of Water Planning and Standards<br>U. S. Environmental Protection Agency<br>401 M Street, S.W.<br>Washington, D.C. 20460 | | | 13. Type of Report & Period Covered<br>Interim<br>14. |

15. Supplementary Notes

16. Abstracts

This document summarizes the physical/chemical properties, toxicological information and environmental fate and effects of Toxaphene, with emphasis on aquatic behavior. From these data a criterion for the protection of aquatic life and human health is developed.

17. Key Words and Document Analysis. 17a. Descriptors

Criteria
Toxicity
Aquatic animals
Aquatic biology
Human ecology
Safety factor

17b. Identifiers/Open-Ended Terms

Toxic Pollutant Effluent Standards
Federal Water Pollution Control Act

PRICES SUBJECT TO CHANGE

17c. COSATI Field/Group

| 18. Availability Statement | 19. Security Class (This Report) | 21. No. of Pages |
|---|---|---|
| Release Unlimited | UNCLASSIFIED | 63 |
| | 20. Security Class (This Page) UNCLASSIFIED | 22. Price |



FORM NTIS-35 (REV. 10-73) ENDORSED BY ANSI AND UNESCO. THIS FORM MAY BE REPRODUCED USCOMM-DC 8265-P74

# CRITERIA DOCUMENTS FOR TOXIC POLLUTANTS

Scientific rationale and criteria developed pursuant to Section 307(a) of the Federal Water Pollution Control Act, P.L. 92-500, 33 U.S.C. §§ 1251 et seq., (1972), for the development and establishment of effluent limitations for toxic substances are set forth in the following chapters.

Section 307(a)(2) states inter alia that a proposed effluent standard "...shall take into account the toxicity of the pollutant, its persistence, degradability, the usual or potential presence of the affected organisms in any water, the importance of the affected organisms and the nature and extent of the effect of the toxic pollutant on such organisms..." Thereafter, having considered these factors, the Administrator is to set an effluent standard for toxic pollutants which provides an ample margin of safety.

In the development of criteria which serve as both the basis and the goal for the establishment of effluent limits, reliance was placed on the toxicity data derived from laboratory studies on a range of organisms including invertebrate, vertebrate, and mammalian test species. These studies provided extensive acute and chronic toxicity data based on feeding experiments for a wide range of aquatic organisms and consumers of aquatic organisms. Environmental studies documenting bioaccumulation in the food web of the toxic material by the food chain organisms and bioconcentration by organisms directly from water provided an important component data base upon which criteria were derived. Appropriate human toxicity data and mammalian carcinogenic studies, where available, were used also in developing criteria.

Aquatic toxicity data generally are obtained by one of two basic methods, the static and flow-through bioassay. The more traditional static bioassay employs a tank in which the test organisms are living and to which a given concentraction of toxicant is added. Any water loss due to evaporation is made up by the addition of fresh water. The flow-through bioassay, which is a more recent development, reflects more nearly the natural conditions. Concentrations of toxic substances are constantly maintained and provide a more accurate test of sensitivity of aquatic species. Water in a flow-through test is replenished constantly through flushing. Comparative results using the static and flow-through bioassays demonstrate that flow-through data yield lower toxicity values for a pollutant than a static bioassay. This fact is demonstrated by comparative studies as discussed in the endrin document. However, most of the data available were developed using static bioassays.

Some toxic pollutants are extremely stable and degrade only slowly or form persistent degradation products. Those pollutants which degrade rapidly pose a less severe long-term hazard unless their entry to the environment is continuous. A parent compound, e.g., aldrin, may rapidly degrade or be altered to a more toxic form, i.e., dieldrin.

Bioconcentration of toxic pollutants is a significant consideration in the development of criterion. The rate and degree of accumulation in an animal and the rate of loss from the animal are factors that help define the potential magnitude of the pollution load problem. As an

example, a pollutant which bioaccumulates presents a hazard both to aquatic systems and potentially to man or other carnivores associated with the ecosystem. To satisfactorily manage a persistent or non-degradable pollutant requires the maintenance of a ceiling for ambient levels in water which will afford protection to the food chain and the consumers of aquatic life (animals including humans). The body burden of toxic pollutants in fish or food chain organisms may have no outward effect on the species but will affect consumers of that food level. As an example, the brown pelican, when feeding on endrin-contaminated fish may die or suffer species depletion through reproductive impairment.

Data on toxic effects of pollutants are not available for all species that may be exposed to the toxic pollutant in these complex societies. Such data would be necessary to ensure protection of the most sensitive species. It is desirable to know the relative sensitivity of a wide variety of species in order to have a better estimate of the sensitivity of the untested, most sensitive species. Because such data are not available on all species, the range in sensitivity of a small number of tested species is used to provide a measure of the range of sensitivity of all species.

The natural aquatic environment includes many kinds and life stages of plants and animals that are intricately interrelated to form communities. Criteria are developed to protect these interrelationships and incorporate aquatic toxicity data for a phylogenetic cross section of organisms as well as

species representative of wide geographic distribution. Chronic studies are an important consideration in establishing criteria and require studies of at least one generation, i.e., one reproductive cycle. Use of an application factor for persistent and bioaccumulated toxic pollutants represents consideration of a safety factor. As discussed in the National Academy of Science publication on water quality (p. 185 of the NAS/NAE Water Quality Criteria -- 1972, GPO-5501-00520), the use of an application factor of 0.01 when applied to acute toxic values is thought to provide an ample margin of safety for certain chlorinated hydrocarbon pesticides.

Ecological importance of an organism is dependent on the role the organism plays within the ecosystem and upon its relationship to the food chain within the aquatic community and to consumers of aquatic life, including man. Thus, toxicity data for the top carnivores in a given ecosystem, as well as economically important species such as trout, salmon, menhaden and shrimp are needed for the development of a protective criteria level. Toxicity data for organisms such as the stonefly and Daphnia are of equivalent importance since these organisms are a food base for higher consumers and are representative of invertebrate species found in most waters of the United States.

Invertebrate species, such as the stonefly and the Daphnia, are an indication of the integrity of the aquatic food chain and their presence may be the controlling factor for the abundance of economically and

recreationally important predators such as trout, bass or pike. While these fish may not directly consume the Daphnia or stonefly or, in fact, even inhabit the same waters, these lower order organisms are representative of the food chain base supporting predators.

Criteria levels, by their nature, are developed to protect aquatic organisms and consumers of aquatic life from direct toxic effect when placed on contact with the toxic pollutants; and, to protect from a more insidious and even greater danger, e.g., chronic effects. Chronic effects take the form of reproductive failures or the poisoning of predators consuming food organisms which have bioaccumulated levels of toxic pollutants as in the case of the brown pelican and consuming endrin loaded fish (see Attachment D, Endrin), and a variety of other physiological effects as discussed in the various documents. Decreases in aquatic organisms or consumers of aquatic life not always are coupled to point source discharges of toxic pollutants at concentrations below acute toxic levels; however, the addition of toxic levels which are not acutely toxic can achieve the destruction or at least disruption of aquatic systems by causing reproduction of failure. Hence, the need for application factors. The relationships between discharges of toxic pollutants and effects on important organisms of economic and environmental importance and consumers of these organisms are well documented in the criteria documents.

An approach to criteria development is to provide ample protection of the test species on the assumption that the response of these species will be characteristic of other associated organisms in the aquatic environment. A number of species have been considered in establishing a criteria

Use of mammalian systems to determine the carcinogenic potential of toxicants found in water follows the same principle as use of aquatic organisms to determine toxicity to fish and other organisms. Carcinogenic substances pose a special hazard to man through environmental exposure. Cancer producing substances may reach man by several distinct pathways.

The following four criteria documents for aldrin/dieldrin, DDT and its metabolites, endrin and toxaphene, represent a survey of the scientific literature documenting the effects of these toxic pollutants to aquatic life and consumers of aquatic life including man. A glossary of terms is provided to define the terms used throughout the documents and will be expanded as necessary when additional documents are added.

TOXAPHENE

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## I. Preamble

Toxaphene (chlorinated camphene) behaves much like other chlorinated hydrocarbon pesticides in the aquatic environment. That is, it degrades slowly, adsorbs on particulate matter and concentrates in living tissues, especially lipids. At one time it was widely used as a piscicide for the elimination of "rough" fish from lakes prior to selective restocking, but its persistence made it unsuitable for such applications.

Toxaphene is highly toxic to both vertebrates and invertebrates. It accumulates in plants and animals and persists for long periods in the aquatic environment. At extremely low levels it has been shown to cause the "broken back syndrome" in fish fry (15). Toxaphene has been associated with the death of birds that feed on fish living in contaminated waters (1).

The purpose of this document is to review available data concerning the properties, toxicity and environmental fate and effects of toxaphene. Based upon the data, a concentration in water of 0.005 ug/l (ppb) is recommended as a criterion level.

## II. Chemical and Physical Properties

Toxaphene is defined as chlorinated camphene (67-69 percent chlorine) and has the empirical formula $C_{10}H_{10}Cl_8$ with an average molecular weight of 414.

Toxaphene (U.S. Patents 2,565,471 and 2,657,164, Hercules) is commercially produced by reacting camphene and chlorine in the presence of ultraviolet radiation and certain catalysts to yield chlorinated camphene with a chlorine content of 67 to 69 percent. The final product is a relatively stable mixture of related compounds and isomers with a mild terpene odor.

A large number of chlorinated compounds is present in toxaphene. A typical gas chromatogram suggests that 30 or 40 principal constituents may exist. The chlorine content in the commercial product is limited to 67 to 69 percent since insecticidal activity peaks sharply in that range.

Infrared absorptivity at 7.2u helps distinguish toxaphene from other chlorinated terpene products such as Strobane. Some tricyclene may accompany the camphene, but less than 5 percent of other terpenes is present (1).

## Table 1

### Physical Properties (1)

| | |
|---|---|
| Physical form: | Amber, waxy solid. |
| Melting point: | 70°-90°C. |
| Solubility: | High solubility in most organic solvents, but greater in aromatic solvents; water solubility is about 0.5 ppm. |
| Vapor pressure: | 0.2-.4 mm/25°; 3-4mm/90°C. |

### Product Specifications

| | |
|---|---|
| Total organic chlorine, % by weight | 67.0-69.0 |
| Acidity, % by weight as HCl | 0.05% max. |
| Drop softening point, °C | 70 min. |
| Infrared absorptivity at 7.2u | 0.0177 max. |
| Specific gravity at 100°C/15.6°C | 1.600 minimum |

When dispersed in a natural water toxaphene tends to be adsorbed by the particulates present or to be taken up by living organisms in which it may be bioaccumulated. Thus, it is seldom found at high levels as a soluble component in receiving waters but can persist in sediments and on suspended solids for prolonged periods.

## III. Toxicological Aspects

Toxic effects resulting from the presence of toxaphene in water have been documented for aquatic organisms representing a wide phylogenetic cross section and a wide geographic distribution. While all test organisms used may not be universally distributed in the waters of the United States, they do represent types of organisms present in fresh, marine, and estuarine systems. Extrapolation from the effects found in laboratory and field tests is the only means for predicting effects of toxaphene on individual organisms and their food chains.

It should be noted that the LC50 values reported for static bioassays are likely to be substantially higher that LC50 values found using flow-through bioassays. Because of a paucity of flow-through data, comparisons of flow-through and static results with toxaphene are not feasible. However, with endrin Earnest in 1970 (76) found a 96-hour TL50 (LC50) of 4.7 ug/l of endrin for Korean shrimp, Palaemon macrodactylus, using a static bioassay and a TL50 (LC50) of 0.30 ug/l using an intermittent-flow bioassay. This may explain why Katz in 1961 (22) reported a 96-hour static TLm (LC50) for endrin of 1.2 ug/l in fresh water for chinook salmon, Oncorhynchus kisutch, while Earnest in 1970 (76) calculated a value of 0.14 ug/l, about one-tenth of the static bioassay value, from freshwater intermittent-flow bioassay data. These data suggest loss of toxicant in static bioassays. Static tests in which dissolved oxygen and toxicant concentrations are

measured periodically are more reliable than those in which these parameters are not monitored. The flow-through bioassays more accurately reflect nature, where "container wall" effects are likely to be negligible and where the volume of water per fish is much greater.

A review of published toxciity values shows that toxaphene is extremely toxic to many fish (16, 20, 21, 22) and to invertebrate species such as the stoneflies (9).

In humans acutely toxic doses of toxaphene produce symptoms that include salivation, spasms of the leg and back muscles, nausea, vomiting, hyperexcitability, tremors, chronic convulsions and tetanic contractions of all skeletal muscles. Most of these effects are the results of diffuse stimulation of the cerebrospinal axis. After lethal doses the convulsions continue until death occurs. Respiration is arrested due to tetanic muscular contractions (5).

A. Microscopic Life

The effects of 17 toxicants on 5 pure cultures of marine phytoplankton were reported by Ukeles in 1962 (8). Toxaphene was the most potent of the chlorinated hydrocarbons tested.* At a concentration of 10 ug/l no appreciable effect was seen with four species; however, a concentration of 150 ug/l was lethal to all organisms. One organism, Monochrysis lutheri, was killed by a concentration of 0.15 ug/l toxaphene but not by a concentration of .015 ug/l (8).

*Other tested substances were: TEPP, Depterex, carbolic acid, Dowacide A, o-dichlorobenzene, Niagara compound 3514, PVP, DDT, Lindane, Nabam, Sevin, Lignasan, Fenuron, Monuron, Diuron, and Neburon.

B. Aquatic Invertebrates

1. Freshwater

Toxaphene LC50 data for several species of freshwater invertebrates commonly found in U.S. waters exposed to toxaphene are found in Table 2.

TABLE 2

LC50 Values for Various Freshwater Invertebrates (Static Bioassays)

| Species | LC50 (ug/l) 24-hr. | 48-hr. | 96-hr. | Ref. |
|---|---|---|---|---|
| Stonefly | | | | |
| *Claassenia sabulosa* | 6 | 3.2 | 1.3 | 9 |
| *Pteronarcella badia* | 9.2 | 5.6 | 3.0 | 9 |
| *Pteronarcys californica* | 18 | 7 | 2.3 | 9 |
| Amphipod | | | | |
| *Gammarus lacustris* | 180 | 70 | 26 | 10 |
| Waterflea | | | | |
| *Daphnia pulex* | | 15 | | 11 |

Sanders and Cope (11) conducted laboratory bioassays with toxaphene to determine immobilization values for two species of amphipods, *Daphnia pulex* and *Simocephalus serrulatus*. Estimated 48-hr. EC50 immobilization values for *S. serrulatus* were 19 ug/l at 60°F and 10 ug/l at 70°F. For *D. pulex* the value was 15 ug/l at 60°F.

It has been demonstrated that the stoneflies, Pteronarcys californica, Pteronarcella badia, and Claassenia sabulosa are extremely sensitive to toxaphene, with 96-hr. LC50 values of 1.3 to 3 ug/l (9).

2. Marine

Schimmel in 1975 (13) found a 96-hour EC50 of 1.4 ug/l for the pink shrimp, Penaeus duorarum, 4.4 ug/l for the grass shrimp, Palaemonetes pugio, and 4.9 ug/l for the American oyster, Crassostrea virginica. Actual toxicant concentrations were measured during exposures. Although few data are available, it is reasonable to expect further testing to uncover more sensitive species.

D. Fish

1. Freshwater

Considerable work has been done on the effects of toxaphene on freshwater fish. The data in Table 3, obtained under various exposure conditions, illustrate its high toxicity to many species widely distributed in fresh waters of the United States.

TABLE 3

Toxicity to Various Freshwater Fishes
(Static bioassays)

| Fish Species | 96-hour LC50 (ug/l) | Reference |
|---|---|---|
| Largemouth bass (Micropterus salmoides) | 2 | 20 |
| Brown trout (Salmo trutta) | 3 | 20 |
| Bluegill (Lepomis macrochirus) | 3.5 | 21 |
| Carp (Cyprinus carpio) | 4 | 20 |
| Black Bullhead (Ictalurus melas) | 5 | 20 |
| Goldfish (Carassius auratus) | 5.6 | 21 |
| Coho salmon (Oncorhynchus kisutch) | 8 | 20 |
| Rainbow trout (Salmo gairdneri) | 11 | 20 |
| Yellow perch (Perca flavescens) | 12 | 20 |
| Channel catfish (Ictalurus punctatus) | 13 | 20 |
| Redear sunfish (Lepomis microlophus) | 13 | 20 |
| Goldfish (Carassius auratus) | 14 | 20 |
| Fathead minnow (hard water) (Pimephales promelas) | 5.1 | 21 |
| Fathead minnow (soft water) (Pimephales promelas) | 7.5 | 21 |
| Guppies (soft water) (Poecilia reticulata) | 20 | 21 |
| Chinook salmon (Oncorhynchus tshawytscha) | 2.5 | 22 |

| Fish Species | | 96-hour LC50 (ug/l) | Reference |
|---|---|---|---|
| Coho salmon (Oncorhynchus kisutch) | | 9.4 | 22 |
| Rainbow trout (Salmo gairdneri) | | 8.4 | 22 |
| Rainbow trout | (53°F.) | 8.4 | 16 |
| Stonerollers (Campostoma anomalum) | (53°F.) | 14 | 16 |
| | (73°F.) | < 5 | 16 |
| Goldfish (Carassius auratus) | (53°F.) | 94 | 16 |
| | (73°F.) | 50 | 16 |
| Golden shiner (Notemigonus crysoleucas) | (73°F.) | 6 | 16 |
| Bluntnose minnow (Pimephales notatus) | (53°F.) | 30 | 16 |
| | (73°F.) | 6.3 | 16 |
| Black bullhead (Ictalurus melas) | (53°F.) | 25 | 16 |
| | (73°F.) | 1.8 | 16 |

Mayer and co-workers (14, 15) in 1974 and 1975 demonstrated the chronic effects of toxaphene on two species of freshwater fish. In one series of tests, young fathead minnows, Pimephales promelas, were exposed for 150 days to concentrations of toxaphene as low as 0.055 ug/l in a flow-through system. At the end of the exposure period the growth of all fish exposed to all levels of the toxicant was significantly reduced, indicating the no-effect level of toxaphene to be less than 0.055 ug/l (14).

In the other series of experiments year-old brook trout, Salvelinus fontinalis, were continuously exposed for as long as 180 days to water concentrations of toxaphene ranging from 0.502 to 0.039 ug/l in a flow-through system. Water temperature and duration of light exposure were altered to correspond to natural conditions. The results of the experiment may be summarized as follows: growth of yearling brook trout was reduced through continuous exposure to 0.288 and 0.502 ug/l for 180 days; concentrations of 0.068 ug/l and higher reduced egg viability; the composition (i.e., the ratio of calcium plus phosphorus to collagen) of the vertebral column of fry was significantly altered during a 90-day exposure; there were marked liver, pancreatic, and kidney pathologies; and finally, the mortality of fish at every concentration tested was significantly greater than in the controls, indicating a no-effect level of less than 0.039 ug/l (15).

Mahdi in 1966 (16) determined lethal concentrations of toxaphene for the stoneroller, Campostoma anomalum; golden shiner, Notemigonus crysoleucas; goldfish, Carassius auratus; black bullhead, Ictalurus melas; and bluntnose minnow, Pimephales notatus in water at 53°F and 73°F; rainbow trout, Salmo gairdneri were tested at 53°F. These tests indicated that the potency of toxaphene increased with temperature. The 96-hr. TLm (LC50) values were below 100 ug/l of toxaphene for all species tested and for fish other than goldfish, ranged from 1.8 to 30 ug/l.

Macek, et al., in 1969 (18) studied the effects of temperature on the susceptibility of bluegills to toxaphene. Their data are presented in Table 4.

Ferguson, et al., in 1965 (17) reported the tolerances of black bullheads, Ictalurus melas, and mosquitofish, Gambusia affinis, from a transect of the lower Mississippi River. The approximate 36-hour TLm (LC50) value for mosquitofish from five main river sites ranged from 10 to 30 ug/l, while a resistant population of mosquitofish yielded a TLm (LC50) of 480 ug/l. Mississippi River data on black bullheads from four sites gave 36-hr. TLm (LC50) values of 3.75, 12.5 50, and 22.5 ug/l. Obviously the more resistant fish could transfer more toxaphene to predators, including man.

Predation by sunfish on toxaphene-contaminated mosquitofish caused mortality to the sunfish (68).

In ethological studies using the Comet strain of goldfish, Carassius auratus, Warner and associates in 1966 (19) showed that behavioral pathology occurred when the fish were exposed to sublethal doses of toxaphene. Effects were observed at levels as low as 0.44 ug/l, which is approximately 1/25 of the 96-hour TLm (LC50) reported by Macek and McAllister in 1970 (20). Behavioral pathology was scored using a complicated system based on a number of parameters, including responses to light stimulus, total movement and habituation to light stimulus.

TABLE 4

24 and 96-hr. TL50 (LC50) Values for Bluegills at Various Temperatures (18)

| Exposure Time | 12.7°C | 18.3°C | 23.8°C | R.I.S.* |
|---|---|---|---|---|
| | TL50 (ug/l) | | | |
| 24 hours | 9.7 | 6.8 | 6.6 | 1.46 |
| 96 hours | 3.2 | 2.6 | 2.4 | 1.71 |

*R.I.S. = Relative increase in susceptibility: The ratio of the TL50 value at the lowest temperature to that at the highest temperature.

2. Marine

The pinfish, Lagodon rhomboides, is an omnivore (73). It ranges from Cape Cod to the Yucatan Peninsula and in many areas is the most abundant species of nonpelagic fish (74, 75). It serves as a link in the food chain supporting such commercially important species as the spotted sea trout, Cynoscion nebulosin; the sailfish, Istophosus americanus; and the gulf flounder, Paralichthys albiguttas. It is also of some importance as bait and as human food (74).

In 1975 Schimmel (13) reported a 96-hour EC50 value for the pinfish, Lagodon rhomboides, to be 0.5 ug/l (measured concentration) in a flow-through bioassay. Chronic exposure of spot, Leiostomus xanthurus, to sublethal concentrations (0.1 ug/l) of toxaphene in seawater for five months under flow-through conditions yielded evidence suggestive of histopathological changes (23). Korn and Earnest in 1974 (69) reported a TL50 (LC50) of 4.4 ug/l for the striped bass, Morone saxatilis.

E. Birds

Keith in 1966 (24) studied groups of five white pelicans maintained on sardines contaminated with each of the following toxicant concentrations: 10 ppm toxaphene, 50 ppm toxaphene, 50 ppm DDT or a combination of 10 ppm toxaphene and 150 ppm DDT. Death occured earlier in those pelicans fed toxaphene at the 50 ppm level than in all other groups.

Toxaphene LD50's have been reported for a variety of birds: young mallards (less than six months of age), 70.7 mg/kg; young pheasants, 40.0 mg/kg; young bobwhite quail, 85.4 mg/kg; sharp-tailed grouse, 10 to 20 mg/kg; fulvous treeducks, 99 mg/kg; and lesser sandhill cranes, 100 to 316 mg/kg (25).

In 1958 a study was conducted on pheasants fed 100 and 300 ppm of toxaphene in their diets for two to three months (27). The 300 ppm level caused a decrease in egg laying and hatchability and in the food intake and weight gain. Both dose levels caused greater mortality in young pheasants during the first 2 weeks after hatching than was observed in the controls.

F. Mammals

Acute toxicity

The acute toxicity of toxaphene has been determined for a number of mammalian species. A comparison of the oral and dermal toxicities of several chlorinated hydrocarbon insecticides in rats under standardized conditions was published by Gaines in 1960 (28). Table 6 presents data from that report. For oral administration, the compounds were dissolved or suspended in peanut oil. For dermal application xylene solutions were used.

TABLE 5

Acute Oral and Dermal Toxicity of Several Chlorinated Hydrocarbons to Rats (28)

| Compound | Oral LD50 (mg/kg) | | Dermal LD50 (mg/kg) | |
|---|---|---|---|---|
| | Males | Females | Males | Females |
| Toxaphene | 90 | 80 | 1075 | 780 |
| DDT | 113 | 118 | - | 2510 |
| Chlordan | 335 | 430 | 840 | 690 |
| Aldrin | 39 | 60 | 98 | 98 |
| Dieldrin | 46 | 46 | 90 | 60 |
| Endrin | 18 | 8 | - | 15 |

The data in Table 5 show that toxaphene resembles DDT in acute oral toxicity to rats. A number of factors influence the toxicity of toxaphene. The route of administration, the solvent used for the tests, and the species must be considered in evaluating the potential hazard.

The data in Table 6 show the variation in toxicity of toxaphene given orally and dermally to several common species by means of different vehicles.

TABLE 6

LD50 Values of Toxaphene to Several Mammals (1)

| Species | Route | LD50 (mg/kg) | Vehicle* |
|---|---|---|---|
| Rat | Oral | 90 | Peanut oil |
| Rat | Oral | 60 | Corn oil |
| Rat | Oral | 120 | Kerosene |
| Mouse | Oral | 112 | Corn oil |
| Dog | Oral | 49 | Corn oil |
| Dog | Oral | >250 | Kerosene |
| Guinea pig | Oral | 270 | Corn oil |
| Guinea pig | Oral | 365 | Kerosene |
| Cat | Oral | 25-40 | Peanut oil |
| Rabbit | Oral | 75-100 | Peanut oil |
| Rabbit | Oral | 250-500 | Kerosene |
| Cattle | Oral | 144 | Grain |
| Goat | Oral | 200 | Xylene |
| Sheep | Oral | 200 | Xylene |
| Rat | Dermal | 930 | Xylene |
| Rabbit | Dermal | >4000 | Dust |
| Rabbit | Dermal | < 250 | Peanut oil |

*The vehicle used to apply a toxicant may influence the rate and extent of toxicant absorption by the animal, whether by oral or dermal or other mode of application. Furthermore, some vehicles may themselves be significantly toxic.

Assuming for the sake of safety that man resembles the most sensitive experimental species (the cat), the lethal dose for a 70 kg adult would be about 2 to 3.5 g. The lethal dose for man has been estimated by others to be from 2 to 7 g (3, 29).

Chronic toxicity

The chronic toxicity of toxaphene was studied by Ortega et al., in 1951 (31) in small groups of rats fed 50 and 200 ppm in the diet. These dietary levels produced no clinical signs of toxicity, inhibition of food consumption or growth rate. Only the livers, spleens, and kidneys were examined histologically. There was no damage to the kidney or spleen but the livers of 3 of the 12 rats that received 50 ppm showed slight changes. Six of the 12 rats fed 200 ppm showed distinct liver changes.

In lifetime feeding studies with rats, dosages of 25, 100, and 400 ppm of toxaphene in the diet were investigated (32). Liver enlargement was noted at all levels.

The kidneys and livers of 2 dogs fed toxaphene at a rate of 4 mg/kg of body weight per day for 106 days showed degenerative changes (33).

Innes et al., in 1969 (34) attempted to cause tumors in mice by stomach tube administration of maximum tolerated doses of pesticides starting at 7 days of age and continuing to 4 weeks of age. From 4 weeks of age until 18 months of age, the chemicals were fed in the diet at levels near the maximum tolerated dose. Toxaphene was not included in that study but the closely related material, Strobane, given at a daily dose of 4.64 mg/kg between ages 7-28 days and at 11 ppm in the diet thereafter caused a higher incidence of lymphomas and hepatomas than was seen in controls.

Toxaphene can change the toxicity of drugs and other chemicals detoxified by hepatic microsomal enzymes and alter steroid metabolism because it induces synthesis of hepatic microsomal enzymes. Dose-response relationships for enzyme induction by toxaphene were measured by feeding various dietary levels to rats for as long as 13 weeks. The lowest dietary level of toxaphene that caused induction of the synthesis of one or more of the three microsomal enzymes studied was 5 ppm. Maximum induction occurred within the first 3 weeks of the feeding period at all levels of toxaphene that caused enzyme induction. After this time the activity was maintained at a constant, elevated level until feeding of the pesticide was discontinued (35).

These results show that dietary levels of 5 ppm and higher could alter the rate at which other chemicals are metabolized.

Similar enzyme induction was obtained with DDT at a dietary level of 1 ppm.

G. Humans

Acute toxaphene poisoning in humans is rare. When toxaphene was first used, four cases of poisoning by ingestion in children under 4 years of age were reported (5). The same report contained a description of severe toxaphene poisoning in adults from accidents or injudicious use of the pesticide. The dosage of toxaphene estimated to have been ingested by three of the people ranged from 9.5 to 47 mg/kg.

Inhalation of toxaphene can cause irritation of the respiratory tract. Warraki in 1968 (47) has described acute bronchiolitis with miliary lung shadows in two men with an occupational history of heavy and prolonged exposure to toxaphene sprays. The threshold limit value for atmospheric levels of toxaphene has been established by the Conference of Government Industrial Hygienists at 0.5 mg per cubic meter of air (48).

## IV. Environmental Fate and Effects

Toxaphene may persist in soil for several months to more than 10 years (62). It has been shown to persist for up to 9 years in lakes and ponds (49, 50, 52).

The fate and movement of a pesticide in and from the soil depend on the following broadly categorized factors: (a) the pesticide characteristics; (b) edaphic considerations; (c) climate; (d) topography; and (e) land use and management. Any of these factors that tend to promote the pesticide's persistence will tend to increase its potential for environmental dispersion (2).

Pesticide movement through or across soil is facilitated by the movement of water. Overland flow is generally more important in pesticide transport than passage through soil. Two processes are involved: (a) pesticide movement while dissolved in water and (b) pesticide movement while adsorbed on soil. Reports of the water solubility of toxaphene have ranged from 0.4 mg/l to 3 mg/l (56).

Bailey and White in 1970 (57) stated that the principal means of pesticide transport within soils are: (a) diffusion in the airspaces of soil; (b) diffusion in soil water; (c) downward flowing water; and (d) upward moving water.

A report by Hermanson et al., in 1971 (58) gives half-life figures on a number of chlorinated pesticides, including toxaphene, in Holtville sandy clay. The half-lives of toxaphene and DDT were calculated to be 4 years.

Johnston, et al. (59), studied pesticide concentrations in tile drainage and open drains in the San Joaquin Valley of California between 1963 and 1965. Toxaphene was detected in 13 of 66 analyses of tile drainage effluents in concentrations varying from 0.13 ug/l to 0.95 ug/l and averaging 0.53 ug/l. Water from surface drains that collected surface and subsurface water was positive for toxaphene in 60 out of 61 samples. Concentrations varied from 0.10 ug/l to 7.90 ug/l and averaged 2.01 ug/l. The predominant residues found

in surface waters were DDT/DDT and toxaphene. The average concentration of toxaphene was higher than that of any other chlorinated hydrocarbon insecticide and it was found most frequently.

Veith and Lee in 1971 (60) investigated the sorption of toxaphene to lake sediments and concluded that within the limits of their analytical system, detectable concentrations of toxaphene could not be leached from sediments by lake water. However, their limit of detection was about 1 ug/l and they carefully concluded, "Also, the laboratory study does not rule out lake waters that may contain ng/liter concentrations of toxaphene due to desorption from the sediments." They also explicitly did not rule out the possibility of the transport of toxaphene from lake sediments through the migration of bottom fauna.

Nicholson et al., in 1966 (61) showed the relative importance of sediment versus solution in the transport of toxaphene, DDT and BHC in Flint Creek, Alabama. Suspended sediment seemed less important to toxaphene and BHC transport than to DDT transport. This suggests a lesser affinity of toxaphene for solid substrates than that possessed by DDT, which is strongly hydrophobic. Support for this contention comes from the fact that toxaphene was frequently recovered in clarified and treated municipal drinking water while DDT was found less often.

Johnson, et al., in 1966 (49) used gas chromatography to study the mechanisms of detoxification. This study shows that toxaphene may persist in a lake for several years after application for fishery management even when detoxification is rapid. All of the lakes studied were shallow and eutrophic. The authors point out that detoxification is accomplished, in part, by sorption reactions rather than degradation, but indicate some evidence based on the gas chromatographic "fingerprint" that toxaphene may be chemically modified.

Johnson and Lew in 1970 (64) determined chlorinated hydrocarbon insecticide residues in fish of the Lower Colorado River system which drains an irrigated agricultural area where insecticides are often used. The following residues of DDT and its congeners, DDT and TDE, and toxaphene were reported in the fat and/or viscera of these fish.

| | | |
|---|---|---|
| Carp<br>Cyprinus carpio | : | DDT, etc., 2.0 - 185.0 mg/kg<br>toxaphene, 50.0 mg/kg |
| Channel Catfish<br>Ictalurus punctatus | : | DDT, etc., 0.7 - 77.0 mg/kg<br>toxaphene, 8.2 - 11.4 mg/kg |
| Sonoran Sucker<br>Catostomus insignis | : | DDT, etc., 7.3 - 46.3 mg/kg<br>toxaphene, 2.8 - 172.9 mg/kg |
| Gila Sucker<br>Pantosteus clarki | : | DDT, etc., 36.2 - 39.5 mg/kg<br>toxaphene, 25.2 - 42.9 mg/kg |

Although the practice of applying toxaphene to lakes for fisheries management has been discouraged, one study of that usage revealed information on bioaccumulation in the hydrosphere under conditions of gross contamination. Terriere, et al. (65), applied toxaphene in Davis Lake, Oregon at 88 ug/l in 1961 and found in 1962 and 1963 that toxaphene was present in water at average levels of 2.1 ug/l and 1.2 ug/l, respectively. They reported a concentration factor of about 500 for aquatic plants, 1000 to 2000 for aquatic invertebrates, 10,000 to 20,000 for rainbow trout, 4000 to 8000 for Atlantic salmon and 1000 to 2000 for lake bottom and mud.

Hughes in 1970 (66) studied biological accumulation and persistence of toxaphene in Wisconsin lakes. When applied to the lakes in fisheries management, toxaphene in the lake water declined to less than the detection limit (1 ug/liter) within 9 to 12 months. However, aquatic fauna, particularly fish stocked in the lakes following treatment, accumulated as much as 19 ug of toxaphene residues per gram of body weight. In general, prey fish accumulated higher concentrations of toxaphene than did predators. Bluegills stocked in Fox Lake about eight months following the last of 3 treatments accumulated 9.4 ug/g in 176 days, after which toxaphene residues began declining until, after 787 days, 0.8 ug/g remained. Two months after fish were stocked, plankton contained 34 ug/g.

Schimmel, et al. (72), studied the bioaccumulation of toxaphene in the ova of the longnose killifish, Fundulus similus, and found that at a measured water concentration of 0.2 ug/l, the concentration of toxaphene in the eggs rose to 0.3 mg/kg in fourteen days (a factor of 1500) and in thirty-two days to 1.1 mg/kg (a factor of 5500).

Lowe et al., in 1971 (67) demonstrated an accumulation of as much as 30 ppm in oysters exposed for 24 weeks to 1 ppb of toxaphene -- a concentration factor fo 30,000. In the ensuing 16 weeks in clean sea water the oysters excreted 90 percent of this load, but retained 3.0 ppm in their tissues -- still 3.000 times the water concentration they had been exposed to originally.

Mayer et al., in 1975 (15) reported that brook trout concentrated toxaphene by factors as high as 76,000 and that the more highly chlorinated isomers were preferentially stored.

Until recently there was no evidence of the existence of toxaphene conversion products or metabolites in crops that had been exposed to toxaphene or in adipose tissues of animals fed toxaphene diets. When toxaphene was extracted either from alfalfa that had been exposed to toxaphene, or from adipose tissue of steers that had been fed toxaphene-treated alfalfa, it had insecticidal activity identical to that of unaltered toxaphene (1). New studies reported in 1974 and 1975 by Casida's groups, however, produced evidence that toxaphene is degraded in vivo (36). They have shown substantial dechlorination in 14 days or less in tissues of rats given toxaphene orally. Although they consider dechlorination essentially equivalent to detoxification, they have not tested the recovered metabolites on insects.

The effects of toxaphene on plankton in treated lakes were studied by Hoffman and Olive in 1961 (7) using a concentration of 100 ug/l. The groups tested were Rotatoria, Protozoa, and Entomostraca.

The first two of these exhibited decreased growth rates, while the population of Entomostraca disappeared in the treated lakes.

Hilsenhoff in 1965 (12) studied the effects of toxaphene on macroscopic bottom fauna in a reservoir and found that an application level of 100 ug/l markedly affected the Chironomidae and Chaoborus populations. Living Chironomidae were absent from samples taken three days after treatment and population recovery was not complete until nine months later. Chaoborus larvae exhibited no immediate effects, but were absent six months later and had not reappeared when the study ended. Oligochaetes showed no adverse effects from toxaphene; their populations actually increased during the eleven-month study period. Chaoborus larvae were the only profundal organisms adversely affected by treatment of the top 30 feet with 100 ug/l of toxaphene. They were eliminated and had not become reestablished two years after treatment. However, these organisms are not strictly profundal and appear to have been killed during sorties into the epilimnion.

Unusually high mortalities were observed in fish-eating birds at the Tule Lake and Lower Klamath Refuges in 1960, 1961, and 1962. The mortalities were reportedly and apparently due to application of large quantities of toxaphene for several years, beginning in 1956, to the agricultural lands immediately surrounding the refuges. Water from the farmlands drains into the marshes. Toxaphene was found in the fish from the marshes at levels of about 8 ppm and in the fat of fish-eating birds at levels as high as 31.5 ppm (51).

## Criteria Formulation

The data presented above establish that toxaphene is a powerful insecticide capable of persisting in the aquatic environment, the soil or animal tissues for long periods, capable of bioaccumulating by factors of as much as 30,000, and capable of producing deleterious effects on fish at levels as low as 0.039 ug/l. Since at least some aquatic organisms can readily bioaccumulate toxaphene, formulation of criteria for this pesticide in natural waterways must take into account the potential danger to terrestrial predators, including man, which consume that aquatic life. However, many important aquatic organisms are exquisitely sensitive to direct poisoning by toxaphene and must themselves be protected.

Sanders and Cope in 1968 (9) demonstrated 96-hour LC50 values from 1.3 to 3.0 ug/l for several species of freshwater stoneflies. The 96-hour LC50 values for largemouth bass and brown trout have been reported as 2.0 and 3.0 ug/l, respectively. Katz in 1961 (22) demonstrated the 96-hour TLm (LC50) for chinook salmon to be 2.5 ug/l. Schimmel (13) reported a 96-hour EC50 of 1.1 ug/l for the sheepshead minnow.

Mayer, et al., in 1974 (15) experimenting on brook trout, found the no-effect level for a six-month exposure to toxaphene to be less than 0.039 ug/l. Similar studies on the fathead minnow (14) showed that

the no-effect level for this species was less than 0.055 ug/l. It is important to emphasize that in neither series of experiments was a toxaphene concentration used which was low enough to have no effect.

Work by Schimmel in 1975 (13) demonstrated the 96-hour EC50 (death) for adult pink shrimp to be 1.4 ug/l. Lowe (23) demonstrated the 48-hour EC50 for spot to be 1.0 ug/l. The 144-hour EC50 for the same species was 0.5 ug/l. Additional work by Schimmel (13) demonstrated 96-hour EC50 values for pinfish to be 0.5 ug/l.

Lowe et al., in 1971 (67) reported that oysters exposed to 1 ug/l of toxaphene concentrated it about 30,000 times in their tissues. Upon transfer to clean water the oysters excreted the toxaphene slowly; excretion was not complete after 16 weeks.

The data set forth above establish the following facts:

(a) Toxaphene in concentrations clustered around 1 ug/l is acutely toxic to fish and to the lower organisms on which they feed;

(b) Toxaphene can be bioaccumulated by factors as high as 30,000 in aquatic organisms, thereby posing a potential threat to other aquatic organisms, to birds, and to mammals (including man) which feed on them.

(c) Toxaphene can be quite persistent in aquatic systems, on soil, and in animal tissues.

(d) Toxaphene can have serious adverse effects on aquatic life exposed to concentrations as low as 0.039 ug/l and perhaps even lower. At higher concentrations these effects become more pronounced, to the point of death.

Acute toxicity has been reported at levels as low as 0.5 ug/l of toxaphene (13). Thus, some damage to aquatic life could be expected at higher levels. At levels of 1.0 to 2.0 ug/l a large number of species are known to be affected. These include stoneflies (9), the sheepshead minnow (13), the black bullhead (16), the pinfish (13), the pink shrimp (13) and the spot (23). An LC50 is the concentration at which half the population dies and can be lowered by other environmental stresses, for instance, by low dissolved oxygen concentration or elevated temperature.

Chronic Criterion: 0.005 ug/l

Several common aquatic species have LC50 or EC50 values in the neighborhood of 1 ug/l; among these are the Stonefly, Claassenia sabulosa, 1.3 ug/l LC50 (9); the sheepshead minnow, 1.1 ug/l EC50 (13); the black bullhead, 1.8 ug/l LC50 (16); the marine pinfish, 0.5 ug/l LC50 (13); and the pink shrimp, 1.4 ug/l EC50 (16). In addition, Lowe (23) reported a 48-hour LC50 of 1.0 ug/l and a 144-hour LC50 of 0.5 ug/l for the spot.

The 96-hour LC50 for the pinfish, Lagodon rhomboides, an organism of wide geographic distribution and of ecological importance in the food chain, has been reported as 0.5 ug/l (13). While the use of an application factor of 0.01 has been recommended by the NAS-NAE (77), its use is especially appropriate in the case of toxaphene because long-term studies with fathead minnows, Pimephales promelas (14), and brook trout, Salvelinus fontinalis (15), have failed to establish a no-effect level. Application of this factor to the 96-hour pinfish LC50 value yields a criterion of 0.005 ug/l.

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# GLOSSARY

Acutely toxic: Causing death or severe damage to an organism by poisoning during a brief exposure period, normally ninety-six hours or less.

Anadromous fishes: Fishes that spend a part of their lives in seas or lakes, but ascend rivers and streams at certain intervals to spawn. Examples are sturgeon, shad, salmon, trout, and striped bass.

Application factor: The ratio of the safe concentration to the lethal concentration as determined for potential aquatic pollutants administered to species of interest.

Bioaccumulation (Bioconcentration): The phenomenon wherein elements or compounds are stored in living organisms because elimination fails to match intake.

Carcinogenic: Producing Cancer.

Catadromous fishes: Fishes that feed and grow in fresh water, but return to the sea to spawn. The best example is the American eel.

Chronically toxic: Causing death or damage to an organism by poisoning during prolonged exposure, which, depending on the organism tested and the test conditions and purposes, may range from several days, to weeks, months, or years, or through a reproductive cycle.

EC50: The concentration at which a specified effect is observed under the test conditions in a specified time in fifty percent of the organisms tested. Examples of specified effects are hemorrhaging, decreased feeding, dilation of pupils, and altered swimming patterns.

Epilimnion: That region of a body of water that extends from the surface to the top of the thermocline and does not have a permanent temperature stratification.

Flow-through bioassay: An assay system in which aquatic species are exposed to toxicants in a constantly flowing system, and where the toxicant is replenished continuously or discontinuously.

Hardness (Water): The concentration of the polyvalent metallic ions dissolved in water. Unually it is reported as the equivalent concentration of calcium carbonate ($CaCO_3$).

Hyperplasia: Abnormal multiplication or increase in the number of normal cells in normal arrangement in a tissue.

Hypolimnion: The region of a body of water that extends from the bottom of the thermocline to the bottom of the water body and is essentially independent of most surface phenomena.

LC25: The concentration of a toxicant that is lethal (fatal) to twenty-five percent of the organisms tested under the test conditions in a specified time.

LC50: The concentration of a toxicant which is lethal (fatal) to fifty percent of the organisms tested under the test conditions in a specified time. It is virtually identical with TLm and TL50.

LD50: The dose of a toxicant that is lethal (fatal) to fifty percent of the organisms tested under the test conditions in a specified time. A dose is the quantity actually administered to the organism and is not identical with a concentration, which is the amount of toxicant in a unit of test medium rather than the amount ingested by or administered to the organism.

Liter (l): The volume occupied by one kilogram of water at a pressure of 760 mm of mercury and a temperature of 4 $^{o}$C. A liter is 1.057 quart.

Methylmercury: Mercury which has been methylated, usually through some biological agent, such as bacteria.

Microgram per liter (ug/l): The concentration at which one millionth of a gram (one microgram) is contained in a volume of one liter. Where the density of solvent is equal to one, one ug/l is equivalent to one part per billion (ppb) or one microgram per kilogram (ug/kg).

Microgram per kilogram (ug/kg): The concentration at which one millionth of a gram (one microgram) is contained in a mass of one kilogram. A kilogram is 2.2046 pounds.

Milligram per kilogram (mg/kg): The concentration at which one thousandth of a gram (one milligram) is contained in a mass of one kilogram. A gram contains 1000 milligrams.

Milligram per liter (mg/l): The concentration at which one milligram is contained in a volume of one liter. Where the density of the solvent is equal to one, one mg/l is equivalent to one part per million (ppm) or one milligram per kilogram (mg/kg).

Milliliter (ml): A volume equal to one thousandth of a liter.

Nanogram per liter (ng/l): The concentration at which one billionth of a gram (one nanogram) is contained in a volume of one liter. Where the density of the solvent is equal to one, one ng/l is equivalent to one part per trillion or one nanogram per kilogram (ng/kg).

Neoplastic: Describing any new and abnormal growth, such as a tumor.

Part per million (ppm): A concentration in which one unit is contained in a total of a million units. Any units may be used (e.g., weight, volume) but in any given application identical units must be used (e.g., grams per million grams or liters per million liters). Where the density of the solvent is one, one part per million is equivalent to one milligram per liter.

Parts per thousand (o/oo): A concentration at which one unit is contained in a total of a thousand units. The rules for using this term are the same as those for parts per million. Normally, this term is used to specify the salinity of estuarine or sea waters.

Piscicide: A substance used for killing fish.

Static bioassay: A bioassay in which the toxicant is not renewed during the test.

Thermocline: That layer in a body of water where the temperature difference is greatest per unit of depth. It is the layer in which the drop in temperature is 1 °C. or greater per meter of depth.

TLm - Median Tolerance Limit: The concentration of a test material at which fifty percent of the test animals are able to survive under test conditions for a specified period of exposure. It is virtually synonymous with LC50 and TL50.

TL50: Synonymous with TLm and virtually synonymous with LC50.

Tumorigenic: Causing or producing tumors.